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OXYGEN IN CARDIAC INFARCTION*

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INFARCTION of the myocardium results from narrowing or occlusion of a coronary artery. The commonest cause of such pathologic changes is atherosclerosis; less frequently syphilis of the aorta produces similar effects when the disease involves one of the coronary orifices, or both. Coronary embolism is rarely responsible. The physiologic disturbances associated with cardiac ischemia vary according to three basic factors: 1) the size of the vessel involved; 2) the rate of closure; 3) the efficacy of the remainder of the coronary circulation in maintaining an adequate flow of blood through the heart muscle.

Obviously, the size of the infarct will depend in large measure on the calibre of the artery affected; a large area of infarction, as a rule, will result in more profound disorder than a small one. The location is of lesser importance. Gradual narrowing, with eventual closure, affords an opportunity for the development of a collateral bed so that the final shut-down is apt to be a less upsetting event. For this reason, occlusion in a patient who has suffered from anginal attacks for a number of years is often accompanied by milder symptoms than in one whose attack of coronary thrombosis is the first evidence of serious trouble. Finally, if atheromatosis in the arteries not immediately involved is slight or absent the myocardium receives enough blood to maintain function during the

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early critical period. In deciding on the advisability or necessity of using oxygen in treatment, an estimate of all of these factors must be made.

The symptoms and signs of cardiac infarction vary widely. The entire episode may be "silent" and discovered only in the course of later electrocardiographic examination; or it may be stormy and followed by evidences of shock. All intermediate grades of severity are observed.

The mechanism of shock following cardiac infarction differs from that seen after hemorrhage or trauma. In so-called "surgical shock," it is the peripheral circulation which basically is at fault. The cardiac output is diminished because of a decreased blood volume and a lowered venous return to the heart. In myocardial infarction, on the other hand, it is primarily the central pumping station that fails.[1] The left ventricle, which is predominantly the site of injury, is unable to eject a sufficient volume of blood to support an effective peripheral flow. The infarct, therefore, results not only in an area of anoxia in the myocardium but also in hypoxia of all the organs and tissues of the body. Passive congestion in the lungs impairs their function with respect to the absorption of oxygen, since the gas does not diffuse readily through edematous alveolar walls. Loss of pulmonary distensibility prevents adequate ventilation and the blood passing through the lungs is incompletely aerated. A vicious circle is initiated which does not tend to correct itself unless aid is given from without.

Indications for the Therapeutic Use of Oxygen

It is not necessary to administer oxygen to every patient with a cardiac infarct. But even though the clinical picture at the outset may appear to be mild, extension of a thrombus or the occurrence of pulmonary edema or an arrhythmia may cause sudden and grave changes. It is therefore desirable to have the required apparatus always readily available on short notice. To give oxygen when in doubt is far better than to withhold it until irreversible changes in the circulation have taken place.

Clinical criteria which indicate the need for oxygen therapy cannot be defined with scientific precision. But the usefulness of oxygen has been demonstrated in the following conditions:

1. Cyanosis.
2. Shock.
3. Severe and persistent cardiac pain.

4. Dyspnea.
5. Acute pulmonary edema.
6. Congestive failure.
7. Certain cardiac arrhythmias.
8. Rising heart rate.
9. Sharp fall in blood pressure.
10. Marked leukocytosis.
11. High fever.
12. Cheyne-Stokes respiration.

A bluish or purple color of the ears, lips and finger tips is a simple and graphic index of oxygen lack; any patient with *cyanosis* is a candidate for relief.

In *shock*, there is an ashen-grey tint to the skin, sometimes, but not always, accompanied by cyanosis. The face and body are bathed in clammy sweat. The extremities are cold. The sensorium may be clear and the patient is often apprehensive and restless. The heart rate is rapid; the blood pressure falls, often to levels below 90 mm. Hg systolic. At the onset, the temperature is normal or subnormal, rising, in the course of twenty-four hours, to fever heights.

Cardiac pain is frequently agonizing and is not relieved even by relatively large doses of opiates injected intravenously. If additional amounts are given, there is danger that vomiting may be induced. Discomfort is due to anoxemia of the heart muscle.

Dyspnea frequently is an early sign of left ventricular weakness and its prompt relief may avert more serious functional disorder, such as *acute pulmonary edema.* When frank *congestive failure* develops there is clear evidence that the damage to the myocardium has impaired the ability of the heart to eject amounts of blood adequate to sustain the circulation. Failure may be due, in part, to a deficient venous return to the heart. Better oxygenation of the blood, and particularly of the heart and kidneys, helps to tide the entire organism over the emergency and to restore circulatory balance.

The onset of certain *cardiac arrhythmias* places an added burden on the heart in maintaining its output. Chief among these are ventricular tachycardia, auriculoventricular heart block, and auricular fibrillation or flutter with rapid ventricular rate. Ventricular tachycardia may terminate in ventricular fibrillation and death. In heart block, Adams-Stokes attacks sometimes are most distressing and occasionally a long

period of asystole or ventricular fibrillation may prove fatal. Premature beats, particularly when they arise in the ventricles, are sometimes forerunners of fibrillation of these chambers.

A *rising heart rate* is a sign of a failing heart and perhaps should be classed as early evidence of cardiac insufficiency. A *sharp fall in blood pressure* often is part of the picture of shock, although it may appear independently of it.

A *high leukocyte count*, above 15,000 per cu.mm., indicates an infarct of large size. In fact, the degree of leukocytosis, in the absence of complications which might modify it, is one of the best early indexes of the severity of the initial lesion. It has been noted that a white cell count of over 25,000 per cu. mm. usually denotes a grave prognosis. Similarly, *high fever*, if due solely to the infarct, is a sign of serious cardiac damage. A rectal temperature over 104° F., like a leukocytosis above 25,000, is cause for concern.[2]

Periodic breathing of the Cheyne-Stokes variety is an expression of hypoxia of the respiratory center. The possible effect of morphine in depressing this center must always be kept in mind as a contributing factor.

These evidences of oxygen lack are all helpful guides; but the final decision as to whether oxygen should be given must be based on clinical experience and judgment.

Method and Technique of Administering Oxygen

The nasal catheter, the various types of mask and the tent have, in recent years, become familiar as friendly instruments of treatment and are no longer regarded as gloomy announcers of the embalmer's visit. It is now rarely necessary to convince the patient and his family of the need for oxygen therapy; more often they expect and request it.

For prolonged therapy, the modern tent is the most comfortable and effective way of administering oxygen. The transparent canopy, the quiet operation and the ability to dispense with ice for cooling represent relatively recent improvements. The optimal concentration required must be gauged according to the estimated need of the individual patient. To relieve hypoxia in the ordinary case of cardiac infarction a concentration of 45 to 50 per cent suffices. With careful attention to the prevention of leaks, this can be obtained with a flow of 12 to 13 liters per minute. Somewhat higher concentrations (60 to

65 per cent) can be reached with an oxygen inflow of 15 to 18 liters per minute. Fluctuations to lower levels at intervals during the course of a day are unavoidable because of the necessity of opening the tent periodically for nursing care and feeding.

The mask is useful particularly when a tent is not quickly available or when relatively high concentrations are wanted. Pure oxygen will sometimes relieve severe pain when a mixture fails; but it should not be given for more than 6 hours at a time because, if inhaled for longer periods, it often produces pulmonary irritation and has been known to cause edema of the lungs.[3] Its use may be resumed, if indicated, after the lapse of several hours. A concentration of 70 per cent can be maintained for an indefinite time without toxic effects.

Administration by nasal catheter is the least effective technique, though the simplest. A humidifier in the circuit is used to prevent drying of the throat. The flow should usually be regulated at 5 or 6 liters per minute, which yields a concentration in the inspired air of about 36 to 38 per cent. At 8 liters, 42 per cent can be attained. When greater concentrations are desired the position of the rubber tube may be tried in the oropharyngeal position. However, this placement is apt to cause irritation and, as a rule, is not well tolerated for long periods. The catheter requires lubrication at frequent intervals and cleansing at least every 12 hours.

Results

That the pain associated with acute occlusion of a coronary artery might be relieved by oxygen was first pointed out by Rizer[4] in 1929. In the following year, Levy and Barach[5] reported a small series of cases in which oxygen therapy appeared to influence the course of the illness in a favorable manner and concluded that in some instances it might be responsible for the saving of life. Other workers[6] soon confirmed these observations and in 1934 the same authors[7] published a second paper in which they amplified their earlier findings on the basis of further experience. The use of oxygen in the treatment of suitable cases of cardiac infarction has become accepted practice.

It is difficult to estimate the effects of this procedure on mortality or on the incidence of complications in view of the diverse clinical picture and because, in order to obtain a series of control observations, it would be necessary to withhold oxygen when the need for it might

be urgent. What at first were impressions have been fortified, however, by the repeated experience of many workers in all parts of the world, so that specific inferences are justified.

1. Oxygen therapy brings about subjective improvement. Pain is lessened or abolished, the heart rate falls, and respiration is slower and less labored. The patient is no longer restless. It is possible to curtail materially or stop entirely the use of opiates and sedatives.

2. The state of shock gradually disappears.

3. Cyanosis is diminished or abolished.

4. The onset of congestive failure may be prevented and oxygen aids in controlling it, after its occurrence.

5. The temperature, when elevated, tends to fall.

6. Cheyne-Stokes breathing, if present, is gradually followed by regular respiration.

7. Of great significance is the fact that interruption of oxygen therapy before adequate readjustment of circulatory conditions has taken place results in recurrence of the symptoms and signs just described. Resumption again proves helpful.

Not all of these beneficial effects are to be anticipated in every case; but not infrequently an apparently hopeless situation shows a sharp turn for the better and improvement can be dated from the first day on which oxygen was given. The occurrence of sudden death while a patient is in an oxygen tent is uncommon, perhaps because ventricular fibrillation is less likely to occur when myocardial hypoxia is reduced. Oxygen therapy does not appear to prevent extension or recurrence of the thrombotic process or lessen the likelihood of emboli; reliance is now placed on the use of anticoagulants for these purposes. But it does lighten the burden thrown on a myocardium that has received a sudden, damaging shock. Thus, during the period of greatest stress, it affords an added margin of safety which may be sufficient to promote recovery.

The length of time that oxygen administration should be continued, as in the case of indications for its use, defies precise definition. It will vary in each patient. A fair average might be given as one week; but much longer residence in a tent is often necessary and may lead to recovery. As improvement occurs, the periods out of oxygen for meals, for bathing and for defecation will give a clue as to how well the circulation is able to carry on in room air. Sudden and sustained increase

in pulse or respiration, the occurrence of cyanosis or the appearance of dyspnea or anginal pain indicate that oxygen is still necessary. Sometimes, premature removal will be followed by the gradual onset of congestive failure, as shown by shortness of breath, diminished output of urine, appearance of edema and the presence of rales at the bases of the lungs. Return to the tent is then necessary. On the other hand, if, after slowly increasing the length of time out of oxygen, none of these signs is noted, the patient may be kept in room air for progressively increasing periods during the day, with return to the tent at night. In the course of several days inhalational therapy is entirely discontinued.

Nothing has been said during this discussion, of other aspects of treatment for the patient with cardiac infarction. It should not be inferred from this omission that oxygen is the only form of aid or indeed, the one which is necessarily the most important. Rest, opiates and sedatives, digitalis or quinidine when needed, the mercurial diuretics, the anticoagulants, dietary regulation and good nursing care all have their place in the regimen. It is the proper admixture which makes for a successful outcome.

SUMMARY

Anoxemia of a portion of the heart muscle and hypoxia of all organs and tissues of the body occur in varying degrees after acute coronary occlusion and cardiac infarction. Oxygen lack is manifested by a group of characteristic symptoms and signs of disordered function. Administration of oxygen by inhalation relieves distress and helps in restoring circulatory balance. In certain instances, its effective use is the crucial factor in saving life.

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ANOXIC EFFECTS ON THE ELECTROCARDIOGRAM PRODUCED BY THE 2-STEP TEST

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PAIN in the chest is a common symptom in many extracardiac diseases and in functional conditions. Usually the pain from such sources as spondylitis, gallbladder disease, hiatus hernia, colitis, peptic ulcer, pneumonia, and pneumothorax can readily be differentiated from angina due to coronary artery disease, and from such diseases as anemia or hyperthyroidism. Occasionally it simulates anginal pain, or the latter may be atypical. In these cases the differential diagnosis is often difficult, since patients with coronary artery disease and angina frequently (37 per cent of 600 patients seen in consultation) appear to be normal upon examination, including a 12-lead resting electrocardiogram. Keefer and Resnik were the first to attribute angina pectoris due to coronary disease to anoxia of the myocardium, and this explanation is generally accepted. They and other authors pointed out the occurrence of RS-T depression in the electrocardiogram during spontaneous attacks of angina and in generalized anoxia. Levy, Bruenn and Barach used quantitative tests based on these changes in the electrocardiogram induced by inhaling 10 per cent oxygen to detect coronary insufficiency. Exercise produces similar alterations in the electrocardiogram in patients with coronary disease, and the "2-step" exercise electrocardiogram was introduced to standardize the effort. The similarity of changes seen in the electrocardiogram in spontaneous angina, during the "2-step" exercise test and, in the anoxemia test, indicates that myocardial anoxia or ischemia is